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A young woman with recurrent spontaneous coronary artery dissection.

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Short title: A young woman with recurrent SCAD.

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A 42-year-old woman was admitted to the hospital with non-ST-segment elevation myocardial infarction. Past medical history revealed that the patient suffered from three spontaneous abortions, migraine episodes, esophageal reflux and asthma. The family history for cardiovascular disease was negative.

On admission the patient was hemodynamically stable. Electrocardiography revealed a sinus rhythm at heart rate of 73/min, incomplete right bundle branch block and negative T-waves in leads II, III, aVF, V4-V6. Blood tests showed elevated levels of high sensitive troponin (895 ng/L; normal (N) < 14 ng/L), creatine kinase myocardial band (612 U/L; N < 170 U/L) and NT-proBNP (137 ng/L; N < 130 ng/L), and normal levels of LDL-cholesterol (2.5 mmol/L; N < 3.0 mmol/L).

Echocardiography showed a normal sized, non-hypertrophic left ventricle (LV) with normal ejection fraction (LVEF biplane 61%) and hypokinesia of the LV inferolateral, inferior mid-ventricular and inferior apical. Coronary angiography (CA) revealed a spontaneous dissection (SCAD) of the distal circumflex artery (Cx, FIGURE 1A).

Due to preserved coronary perfusion and no persistent or recurrent angina, the patient was conservatively treated with dual antiplatelet therapy (aspirin for life time and ticagrelor for 12 months) and beta-blocker (bisoprolol).

As probable causes of SCAD the following conditions were considered: fibromuscular dysplasia (FMD), unclear hereditary vasculopathy, systemic inflammatory and connective tissue disorders[1,2]. After a broad diagnostic work-up, the diagnosis of FMD was based on the radiologic findings (FIGURE 1B). The patient was discharged after 5 days.

A few hours later the patient was readmitted with an anterior ST-segment elevation myocardial infarction (FIGURE 1C). The emergency CA revealed a new SCAD of the middle to distal left anterior descending artery (LAD, with TIMI II/III flow; FIGURE 1D). The

patient was qualified for conservative therapy with nitroglycerin i.v. and heparin i.v. (in therapeutic range) added to the previous pharmacotherapy.

Cardiac magnetic resonance imaging revealed transmural late gadolinium enhancement with microvascular obstruction (MVO) and concomitant edema in the LAD territory and a small old infarct scar in the Cx territory; the LVEF was 39% (FIGURE 1E and 1F, *supplementary material, cine-loop*).

After an interdisciplinary consultation, the patient was anticoagulated with phenprocoumon for 6 months and concomitantly received aspirin (lifelong), bisoprolol, lisinopril, rosuvastatin and pantoprazole. Besides an anemia due to recurrent menorrhagia no other clinical problems occurred during the 2 years follow-up. Control echocardiography showed a dilated LV with mildly reduced LVEF (biplane 45%).

SCAD is responsible for about one third of acute myocardial infarctions in women under 50 years [1,2]. The prognosis following SCAD seems to be good, as it usually heals completely over a few months, although a significant recurrence rate is observed [1,2]. In patients with preserved flow in the coronary arteries a conservative management is suggested, followed by a period of prolonged inpatient monitoring [1-4]. The pharmacotherapy and its duration in SCAD remains controversial and the benefits from antiplatelet, anticoagulant and statin therapies are still a matter of debate [1,4]. Assessment for underlying causes is recommended, including screening for extra-coronary arteriopathy such as FMD, however in some patients no underlying definitive diagnosis can be made at the time of SCAD [1,2,5].

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References:

1. Adlam D, Alfonso F, Maas A, et al. European Society of Cardiology, acute cardiovascular care association, SCAD study group: a position paper on spontaneous coronary artery dissection. Eur Heart J. 2018; 39: 3353-3368.
2. Hayes SN, Kim ESH, Saw J, et al. Spontaneous Coronary Artery Dissection: Current State of the Science: A Scientific Statement From the American Heart Association. Circulation. 2018; 137: e523-e557.
3. Theodoropoulos KC, Hussain R, Palmer ND, et al. The use of multimodality imaging in the diagnosis and management of spontaneous coronary artery dissection and intramural haematoma. Kardiol Pol. 2020; 78: 467-469.
4. Saw J, Mancini GBJ, Humphries KH. Contemporary Review on Spontaneous Coronary Artery Dissection. J Am Coll Cardiol. 2016; 68: 297-312.
5. Toggweiler S, Puck M, Thalhammer C, et al. Associated Vascular Lesions in Patients With Spontaneous Coronary Artery Dissection. Swiss Med Wkly. 2012; 142: w13538.

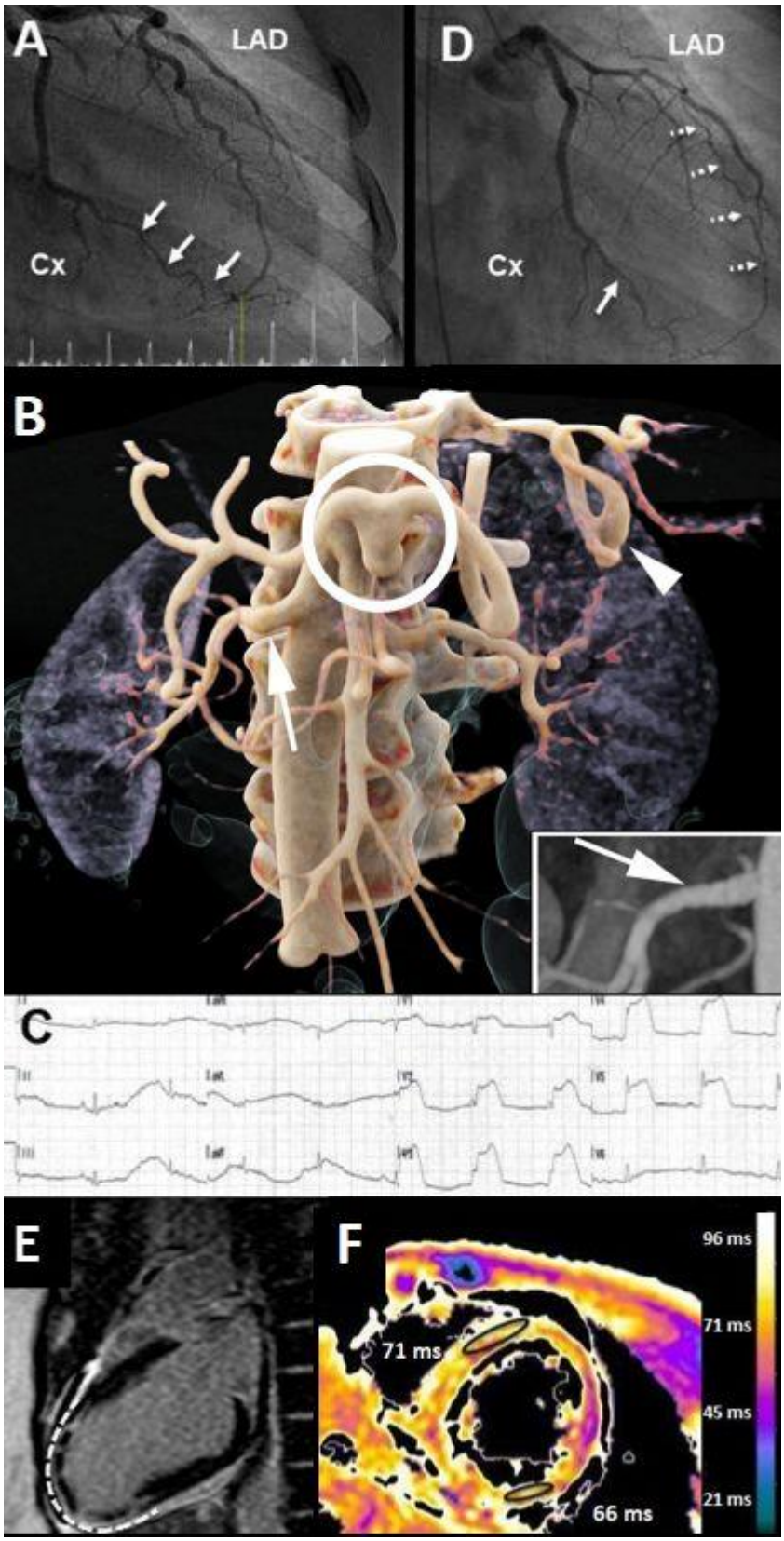
6. 

Figure 1.

A- Coronary angiography on admission: spontaneous dissection of the 1st marginal branch of the circumflex artery (*arrows*);

B- Extracoronary abnormalities. Computed tomography angiography of the abdomen and pelvis: an ectatic coeliac trunk (*circle*), an aneurysm of the distal splenic artery (*arrowhead)*, ectasia of the left iliac internal artery (*not shown*) and a "string of beads" appearance of the right renal artery (*arrow* and *zoom*);

C- 12-lead electrocardiogram with ST-elevation in all anterior wall leads (V1-V5);

D- Emergency coronary angiography: a new spontaneous dissection of the left anterior descending artery (*dashed arrows*) and a residual dissection in the distal circumflex artery (*arrow*);

E, F- Cardiac magnetic resonance imaging showing transmural late gadolinium enhancement in the anterior and inferior apical segments of the left ventricle with microvascular obstruction (E, 2-chamber view, *dashed line*) and edema in all midventricular segments except anterolateral (F, T2-mapping, short axis view).

Abbreviations: Cx, circumflex artery; LAD, left anterior descending artery.